ON HEAT STROKE

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Edinburgh.

April 1909.

HEAT STROKE.

Introduction.

Recently the subject of heat stroke has awakened considerable interest. Although one of the oldest of recognised diseases and a fairly well defined clinical entity, the causation of the condition has not been definitely settled, and recently has given rise to much speculation and several theories as to the mode of production of "sunstroke" and heat stroke have been suggested.

In 1898 Sambon (Bur,I. March 19, 1898) first suggested that the condition was an infectious disease. Although this theory has received some support it cannot be said that a convincing case has been made out for its general acceptance and most recent writers on the subject have repudiated the theory.

Another suggestion and one which seems to be to some extent supported by practical experience is that the condition is due not so much to the ordinary heat rays of the sun as to the actinic or chemical rays.

Colonel Maude R.A.M.C. first suggested this as far back as 1885 and also pointed out that the liability to heat stroke could be diminished if a red or yellow lining was worn under the head covering and coat the reason being that the actinic rays are intercepted by the red or yellow lining.

The question is one worthy of careful study and in the present thesis I have endeavoured to give an outline of the recent developments on the subject together with an account of my own experiences of the condition.

It is first of all necessary to understand clearly what is meant by the expression sun stroke or heat stroke. Fayrer in his book on tropical diseases divides the condition into three classes:

1. Simple Syncope from Exhaustion caused by heat.
2. A condition analogous to shock due to the action of the direct rays of a powerful sun on the brain and cord, the nerve centres especially the respiratory are affected, respiration and circulation rapidly fail and death may result. Recovery is infrequent, though not always perfect.
3. Over-heating of the whole body, blood and nerve centres either from direct exposure to the sun's rays or more frequently to a high temperature out of them, causing vasomotor paralysis and intense pyrexia. Respiration and circulation fail and asphyxia follows.

Recovery frequently occurs but is often incomplete owing to structural changes in the centres giving origin to a variety of symptoms indicative of lesions of a grave character.

Rogers (Journal of the R.A.M.C. Jan 1908 believes that under the terms Heat exhaustion, Sunstroke, Heat stroke and Siriasis two broadly different conditions are included. First - Syncopal attacks due to exposure to the direct rays of the sun or to hard labour during great heat, e.g. in the stokeholds of Red Sea or Persian Gulf steamers.

In these cases there may be no marked elevation of body temperature and if properly treated, recovery is the rule with or without some permanent mental injury.
Second - true heat stroke with hyperpyrexia and acute pulmonary congestion coming on very suddenly usually without any actual exposure to the sun's rays. Such cases only occur under very trying atmospheric conditions either excessive dry heat or lesser degrees of moist heat. This is true heat stroke.

Duncan A. (Journal of Tropical Medicine p. 101,vol v.) describes the varieties of Heat stroke as follows:-

A. Heat collapse.

B. Heat stroke.

(a) direct heat stroke or sunstroke proper.

(b) indirect heat stroke.

A. Heat collapse. The patient suddenly turns giddy and falls. Skin moist and cool. Breathing hurried but never stertorous, pulse small and soft, pupils dilated, temperature at or below normal. No loss of consciousness, and recovery the rule.

B. Heat stroke.

a. Direct heat stroke or sunstroke.

There are several forms:

1. Occurs in persons unaccustomed to marching and attacks them specially when the air is moist. There is violent headache and oppression followed by convulsions, loss of consciousness, difficult respiration, small and irregular pulse and often incontinence of urine.
2. Is characterised by excessive sweating, pallor, cyanosis, shallow breathing, injected eyes, swollen veins and partial collapse without complete unconsciousness. Revival occurs under proper treatment.
3. In this form, no fatigue is complained of, but the patient is thirsty and suddenly falls forwards comatose. The coma may last 24 - 36 hours and end in death.
4. After exertion and exposure to the sun a racking headache sets in. This becomes intense and finally agonising. Great intolerance of light ensues, followed perhaps in 48 hours by unconsciousness. If death does not occur the intense pain in the head may last from 6 or 8 weeks unrelieved by any drug but there may be slight evening remissions. It then gradually abates.

5. Indirect heat stroke. This is the syncopal form occurring not in the open but in the hot house or bungalow. Duncan finds it the most frequent type. At the onset the skin becomes pale, there is nausea, colic and incontinence of urine. Convulsions now follow to be succeeded by cyanosis, dyspnoea and insensibility. The breathing is stertorous, the pupils contracted and the body temperature may reach 108° F. to 110° F. remaining high post mortem.

The standard text books all recognise two forms of the condition:

Heat exhaustion and Heat stroke.

and there is general agreement that the conditions are essentially different.

In Heat Exhaustion the skin is moist, pale and cool, the breathing is easy though hurried, the pulse is small and soft, the senses remain entire, whereas in heat stroke there is usually unconsciousness and pyrexia.

True heat stroke or siriasis may be defined as an acute disease characterised by hyperpyrexia, profound coma and intense pulmonary congestion.

Heat stroke is universally considered as the effect of exposure to excessive heat but opinion varies greatly as to the way in which elevated temperature acts in producing the peculiar symptoms of the disease.

Some authors attribute the condition to a gradual heating of the blood to a degree incompatible with the maintenance of the nervous function.

Others believe that heat paralyses the centre or centres which are supposed to regulate the disposition of bodily heat and thus causes the hyperpyrexia and other symptoms of the disease. This paralysis according to some is supposed to cause a greater heat production. According to others it causes retention of heat.

Little considered heat stroke to be the result of pressure exerted upon the cerebro-spinal matter by the heat expanded cerebro-spinal fluid.

Baxter and Züber thought that the great rise in the bodily temperature depended upon interference with the heat loss as a result of suppression of cutaneous perspiration.

Laveran suggested that the cause of the disease might be perhaps paralysis of the ganglia of the heart (Laveran Bulletin de'l'acad de Med. de Paris 1894). Vallin's theory was that the condition was due to a sudden rigidity of the left ventricle and diaphragm owing to a coagulation of the myosin.

Senfleben (Berliner Klinischer Wochenschrift June 24, 1907, p. 775) ascribes the malady to disorganisation of the blood and accumulation of urea.

Bauer thought that the main factor was an increased liberation of carbonic acid in a blood already saturated with gas.

Smart attributes the symptoms to a deficiency in the serum of the blood from long continued profuse sweating.

These explanations however conflicting are all based on the idea that heat is ths sole efficient cause of the disease. The idea that siriasis is due to excessive heat is of course derived from the fact that it occurs during the hottest season of the year - and there is no doubt that this is one of the most important factors. But it is not the only factor. In one locality the condition may be extremely prevalent, in another it is totally absent though the region may be quite adjacent and quite as hot.

Again its ravages in the same locality but in different years vary immensely and quite irrespective of heat.

These arguments have led some authors to suggest a theory of causation independent of heat.

Colin and de Santi have considered heat stroke as a form of malaria. Long before them Dr. Chevers and other Anglo-Indian surgeons who have given us the best descriptions of the condition had remarked on the probability of sudden attacks of the disease being caused by "malaria in a concentrated form." The idea that malaria was a cause of sun stroke probably sprang from the fact that those suffering from malaria are most liable to the disease, or again from the fact that pernicious malaria may closely simulate it, but it is untenable because in many regions where malaria is most prevalent heat stroke is unknown and

on the other hand it is greatly prevalent in places which have no malaria.

Moreover in heat stroke the spleen is not enlarged.

The belief that heat alone could not account for heat stroke has often been expressed by those who were able to observe the disease in its habitat.

Sambon, the principal advocate of the microbic theory, which is also supported by Sir Patrick Manson and others, bases his principal arguments on the following points:

1. Heat stroke prevails only in low-lying or sea coast districts and the valleys of certain rivers and is never found above 600 feet elevation.
2. Its distribution is capricious and irrespective of the atmospherical temperature curve, and
3. Attacks occur mostly at night when the air temperature is not at its highest point, for which reasons they assert that it cannot be caused by heat alone.

Major Leonard Rogers (Journal of the R.A.M.C. Jan.1908) examined the record of cases of heat stroke in the British Army in India for three consecutive years and his results disproved the above statements. It was found that an exact relationship existed between the meteorological conditions and incidence of sun stroke.

An important point brought out in these observations is the relationship of atmpspheric humidity to sunstroke.

It was found that heat stroke occurred at a much lower mean temperature when a high degree of moisture was present.

The alleged capriciousness in the occurrence of heat stroke cases entirely disappears if the degree of moisture in the air as well as the actual temperature is taken into consideration.

If a high degree of moisture is present interfering with free evaporation of perspiration then the hyperpyrexia of heat stroke may ensue with a maximum air temperature of just about the blood heat.

With a drier atmosphere, the body cooling mechanism may fail when the maximum air temperature exceeds that of the body.

A knowledge of these facts according to Rogers will allow of the occurrence of this dangerous affection being anticipated and measures taken for the early detection and prompt treatment which alone will save the lives of the vast majority of sufferers.

Another interesting report on the relationship of humidity of the atmosphere to sunstroke, is that by Jardine (Clin.Modern N. 22-24 an XII) He discusses his experience during 1905 in regard to cases of sunstroke at Florence.

During that year which was marked by a sudden access of great heat---With high aqueous vapour tension and marked electric state of the atmosphere, the number of cases of sunstroke was considerably above the average.

The great mortality occurred at the beginning of the heat wave before people had had time to get acclimatised or to take proper precautions.

This relationship of the atmospheric moisture to sunstroke is however not borne out by my own experience as will be seen in the cases appended. The rainfall in Dera Ismail Khan averages only six inches per annum and the climate is intensely dry. Yet I found that out of a regiment of Moplah Sepoys who came to Dera Ismail Khan from Malabar (which has a humid climate) twenty-four were attacked with heat stroke on their first day's march.

The curious feature therefore was that these Moplahs coming from a hot humid climate suffered so severely in an extremely dry one. The station they had come from had at this time of the year (September) a higher temperature than Dera Ismail Khan.

In these cases it may also be that another factor comes into play and that is acclimatization. It was first pointed out by Fayrer that acclimatization has some influence in conferring toleration.

Fresh arrivals in the tropics are more prone to suffer than those who have become accustomed to the climate. It is well known that a native can bear an amount of sun on his bare head and naked body with indifference, almost pleasure, that would rapidly prostrate a European.

The question of acclimatization has therefore also to be taken into consideration in my cases. The natives had been accustomed to the humid climate of Malabar and the change to the dry climate of Dera Ismail Khan told on them.

With reference to the actinic theory of sunstroke first suggested by Colonel Maude, it cannot be said to have been definitely established and has been subjected to severe criticism in a paper by Lieutenant Colonel Simpson in the Journal of the Royal Army Medical Corps 1908 page 441.

This theory has, however, in its favour that practical experience to some extent confirms it.

A. Duncan, in the Journal of Trop. Medicine 1907, p. 83, says:

> "There can be no doubt that this actinic theory of sunstroke is the correct one. All soldiers' helmets should be given out to them lined with orange red covers."

Those who have adopted this headgear have certainly found it effective in warding off heat stroke.

The pathology of the condition has been made the subject of a special study by Lambert and Van Dieson (Medical Record July 4, 97) After a study of over eight hundred cases of insolation these observers found that heat alone is not sufficient to explain all the clinical and pathological observations. The prodromal symptoms of sunstroke are

those of acute functional disturbance while the later symptoms much more serious, point to grave changes in the blood and all the nerve centres especially those of the latter which control the thermic mechanism of the body.

Van Gieson examined the brain and cord in several of Lambert's fatal cases and found universal presence of acute degeneration of the neurons of the whole neural axis. In the cerebral cortex and cerebellum the cells showed the same degenerative changes; the cells of the spinal cord were not so extensively involved. A toxic agency of the symptoms of insolation seemed to be shown by the changes found in the ganglion cells. The experiments by Vallin would tend to show that coagulation of the albuminoid bodies occurs, the toxaemia would thus occur as a result of arrested metabolism. The blood is dark though fluid and the corpuscles are crenated.

In the hyperpyrexial form leucocytosis and degeneration of the red corpuscles may also be noted.

Extravasations in the peripheral tissues are often found and the body undergoes rapid putrefaction.

According to de Santi heat stroke is in all cases characterised from a pathological point of view by arrest of the heart but dependent on different causes.

These may be classified as arising from intoxication by the products of muscular effort from asphyxia, from a malarial infection called into activity by fatigue or

heat. In the first form, that of intoxication by the products of muscular exertion, the victims are chiefly among soldiers unaccustomed to the fatigue of a march. The attacks occur when the temperature is high and the air is calm and humid so that cutaneous evaporation is small.

Cagicol and Lapierre (Montreal Clinique '98) found a micro-organism in the blood of patients suffering from heat apoplexy and regarded it as the specific cause of the disease. It is linear, incurved and slightly constricted in the middle. Viewed in the blood it is from 2 to 2.5μ long and 0.5μ thick, in cultures it is somewhat larger. It presents filaments, is slightly motile but possesses no cilia, stains easily with aniline colours, but not by Gram's method.

There are free spores in the cultures as well as in the rods. It is aerobic, does not cause fermentation in sugars and does not give rise to Indol.

These findings have not however been confirmed by later workers and so cannot be accepted yet.

The following cases are of interest as they present some features outside the usual experiences of heat stroke.

Case 1. Major F. sent for me on June 3rd 1906 complaining of persistent dull headache, sleeplessness and depression. T. 101° Pulse 90. He was a thickset, short necked man of 45, with a florid complexion, a lifelong teetotaller and of regular habits. All his previous service had been in Madras. He had been more or less ill for a week, his chief trouble being sleeplessness. He said he had slept only at intervals of an hour or two in a sitting posture as any attempt to lie down caused his head to throb unendurably. He complained very much of the heat. He had never had any similar trouble before coming to Dera Ismail Khan.

Treatment - Saline purgatives, milk diet, Quinine sulphate qr. X daily. He was also given chloral to induce sleep and he was ordered to be removed to a cooler climate but could not arrange to start immediately. The next day his morning temperature was 100.6°F. and evening temperature 100°. General conditions as before. On June 5th I was sent for in the early morning. Patient had spent a very bad night, walking up and down on the flat roof of his bungalow. He was cyanosed and very restless. T. 102.8° Pulse 100. He was intensely nervous and had a feeling of impending death, so much so that he made a hasty will and

wrote farewell letters to his people. I applied ice bags to his head, gave him a dose of calomel gr.V. and a hypodermic injection of strychnine and had abundance of cold water poured over him from the 'bhistes' masak' or leather water bag.

He was perspiring profusely. By mid-day his condition was much improved. He had slept four hours and woke up much refreshed, T.100° pulse 92, rather full but regular. Travelling by day was very dangerous at this season of the year, but at 6 p.m. he started off to drive some 30 miles to the nearest hill station. The servant who accompanied him gives the following history. Fifteen miles out at 8.p.m. Major F. stopped the Tonga and insisted on alighting as he had great difficulty in breathing. Iced cloths were applied to his head and after an interval he resumed his journey.

At the foot of the hill 9.30 p.m. he was again seized with similar symptoms and died quite suddenly before any assistance could be given.

The body temperature four hours after death was 106° and the face so congested as to render the features unrecognisable.

The peculiar features of this case are:

1. Long residence on the plains with no previous trouble on account of heat.
2. Teetotaller always.
3. Long premonitory symptoms.

Case 2. July 3. 1906. Mr. J. aged 26 arrived from outpost duty 40 miles by road travelling at night. He remarked that the station was cooler than the outpost he had just left.

He had been feeling unwell for 3 days past, running a temperature of 100° to 101°. He had no appetite and perspired freely, his bowels were not constipated. He consulted me at 2 p.m. a most unusual hour for Europeans to be abroad. I noticed he was slightly incoherent and much flushed. He complained of occipital headache, great depression, dizziness and flashes of light before the eyes. Temperature at this time was 104°F. Pulse 110 full and bounding. I put him on a grass bedstead in my house and had large quantities of cold water poured over him at once.

Ice bags were applied to his head. He became semi-conscious almost immediately and his temperature rose to 106°F. He was in a critical condition, water was constantly poured over him and a hypodermic of strychnine injected. In about 15 minutes his temperature dropped to 103°F. he gradually recovered consciousness but remained in a dazed condition. That evening his temperature was 101° I arranged to send him away immediately to the hills under the care of a brother officer, as a second syncopal attack would have certainly been fatal.

He remained in a low, dazed condition on the journey to Murree and although his temperature never rose again to over 101° he recovered very slowly from the cerebral condition. He was eventually invalided home suffering from delusions but was physically in good health.

Case 3. Mr. A. aged 20, lately out from home, healthy and athletic. I was called by his servant at 2 p.m. and found him quite delirious. He recognised me at intervals only. Finding his temperature 104° and condition serious I placed him immediately in a cold bath and had large quantities of cold water poured over his head.

The effect was remarkable. In less than 10 minutes he was perfectly rational. Temperature 100°F. He said he felt rather tired and expressed a desire for sleep. The servant had come for me on his own initiative as Mr. A's attack was so sudden in onset, that he had not even time to call for help.

The next day he had so far recovered as to dine in mess. He left for Murree two days later in good health which was maintained. No untoward symptoms have resulted.

Cases 4 to 12. June 29th 1906.

Twenty Sepoys of various castes started to relieve an outpost at 9 p.m. They formed the guard of an ammunition waggon which broke down at midnight. This necessitated off-loading, involving tremendous exertion, the night temperature being 100°F. Nine of these men (four Sikhs, three Pathans and two Dogras) collapsed during this process and became unconscious. Medical aid was not at hand, but their companions carried them to an adjacent house and laid them round the well. Cold water was procured ~~poured~~ in large quantities and thrown over them. In the early morning four of them

were so far recovered as to march back seven miles to the station. The others were all well in a day or two.

Case 13. A. Singh-Sepoy, aged 22. Collapsed on parade at 7 a.m. There had been no previous symptoms. He was carried to hospital in a perfectly conscious condition. T. 107° and a running pulse of 120. Respiration 40 to the minute, quick and shallow. He was placed in an ice pack but the temperature was only lowered to 104° and quickly rose again. Every effort was made to induce perspiration as his skin was very dry. He suffered much from intense headache and had violent cramp in both legs. Hyperpyrexia (T.107°F.) continued even when he was placed in a bath and the temperature lowered by ice. His temperature rose to 110°F. and he died an hour later. The interesting points in this case were:

1. Persistent dryness of the skin.
2. The patient had never touched alcohol.
3. The patient never completely lost consciousness in spite of the pyrexia. No autopsy was possible owing to the caste prejudice.

Cases 14 to 37. Sept. 26th 1906. These were the cases of the 24 Moplah Sepoys alluded to previously. They came from Malabar which has a humid climate and had borne with immunity previous high temperatures but collapsed on their first march in the very dry country of Dera Ismail Khan.

All these Sepoys recovered in the course of a day or

two and none of them suffered any bad after effects. Cases of heat stroke of a mild nature were of daily occurrence in the ranks of this regiment during its first month's sojourn in a dry climate.

At this time of the year Europeans as well as natives in Dera Ismail Khan pursued their ordinary avocations as the temperature was by no means high and the hot weather was considered over.

Case 38. December 1906. Mr. E. a thin anaemic subject had suffered one year previously from a "touch of the sun" in Madras. He had three months of the intense dry heat of Dera Ismail Khan and stood it as well as the rest of us. He remained well until December when he suddenly collapsed from the heat during a mid-day march at the Pindi manoeuvres. The sun certainly has some power even in December but it is not such as to cause apprehension of heat stroke. Mr. E. had been on the march for ten days previously.

This was a typical case of heat exhaustion of the syncopal type. His skin was cold and pale, pulse feeble and rapid, temperature 101°F. respiration quick and shallow. He was carried to the hospital and treated by stimulants, ammonia to the nose, strychnine hypodermically, brandy was also given and a saline purge when consciousness was re-established.

Patient continued unwell for a week running a temperature of 100° - 101°F. and having no appetite. His

mind was not clear and he had some loss of memory. Subsequently for at least six months he could not bear the slightest exposure to the sun's rays, and a year later while on leave in England he had a mild attack of sunstroke one hot summer's day.

My experience in the N.W. teaches me that natives who have never tasted strong drink and Europeans who are total abstainers, are equally liable with those that take alcohol to heat stroke.

Should the Roza (Mahomedan Fast) occur during the hot weather there is always a large increase of cases in Upper India. This tends to show that underfeeding and lowered vitality are the chief predisposing causes. Alcoholism is a predisposing cause only in so far as it lowers the vitality.

It is generally recognised that over exertion tends towards heat apoplexy and troops are moved as little as possible;during the hot weather marches are always accomplished by night.

The following cases are typical ones met with in Assam. Silcher, Assam has an average rainfall of 160 inches per annum and is adjacent to Cherraponjee with a rainfall of 424 inches, by far the wettest district in the world. The temperature in Silchar averages 94°F. at the hottest time of the year. Here natives do not wear pugris and many coolies and Europeans work all day in the tea gardens. Heat stroke, however, is not uncommon, but the cases are absolutely different from those met with in Upper

India. Definite symptoms characterised by sudden onset, and initial high temperature are always present and the malady runs a course much less severe, but the after effects are often serious and long continued. Permanent mental derangement often may follow the mildest cases of heat stroke in this part of India.

There is no doubt that alcoholism markedly predisposes towards heat stroke in Assam.

Case 1. Mr. S. a tea planter, aged 50. A hard drinker and of irregular habits, but well accustomed during 20 years to ~~some~~ sun exposure at all seasons.

On June 17. 1908 he came to Silchar by river steamer. This is the usual method of recuperation adopted by planters after fever. Mr. S. had suffered from a non-malarious fever for one or two days and was much depressed. Ordinarily he had a very cheerful disposition. I was called to see him at the Dak Bungalow. I found him in a condition of collapse, breathing stertorously, his face was flushed and much congested. There was relaxation of the sphincters, pulse very rapid, temperature 106°F. pupils contracted.

He was very evidently in a critical condition. He had been drinking heavily for some days, principally brandy and champagne. I injected strychnine and ice packed him. He regained consciousness rapidly but was extremely feeble, complained of violent headache and was unnecessarily

worried over his affairs. I advised him to postpone his river trip, but feeling better the next morning and having eaten a good breakfast he resumed his journey. The heat on the river was intense that day in spite of the heavy rain. His servant wired for me at 8 p.m. from 30 miles away and travelling by train I reached him at 9.15 p.m. I found him in extremis, in fact he died almost immediately. His temperature was at that time 110° F. Rigor mortis was very sudden, the blood remained fluid, the face and neck were swollen and livid, the pupils widely dilated. The whole body felt pungently hot and decomposition set in almost at once.

Case 2. Mr. B. was an abstemious man, aged 36. He was obliged to spend the great part of his time travelling in the hot weather. He complained very much of the heat even when it was not unusually severe. The least exertion caused him much discomfort, such as severe headache, rushing of blood to the head and dizziness. On July 20th 1908 he was found in a railway carriage in an unconscious condition, evidently suffering from heat stroke. Medical aid was not available but the native station master poured cold water over him and placed him beneath a punkah. He soon recovered and was able to return to Silchar that night. He was afterwards quite unable to bear the least exposure to the sun and even after three months leave in the hills complained of the heat, severe headache and inability to concentrate his mind on his work.

It will be seen from the foregoing that cases of heat stroke while agreeing in some main points, differ widely in onset course and sequelae.

The cases met with in the N.W.Frontier were initially more severe than those in Assam, but most of the patients recovered completely and had no after effects such as intolerance of heat, eye trouble or untoward mental symptoms. As regards the treatment I have used various methods according to the indications of each case.

Among the drugs given may be mentioned Antipyretics, Salicylates, Calomel, Salines and Saline infusions, stimulants, brandy strychnine and digitalis.

Locally, ice packs and ice bags, creosote applied to the axillae to induce rapid perspiration, alternate hot and cold treatment by warm blankets and iced baths, and most important of all, abundance of cold well water poured over the whole body from the bhisti's mashak. In my experience by far the best results were obtained by this last method, thus proving once again that the simplest treatment is often the most effectual.

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